October 1983:616–24

Implications of "Reciprocal" ST Segment Depression Associated With Acute Myocardial Infarction Identified by Positron Tomography

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This study was performed to determine whether patients with myocardial infarction with apparently reciprocal ST segment depression exhibit abnormal metabolism in zones distant from the primary zone of infarction. Positron emission tomography was performed after the intravenous injection of carbon-11 palmitate in 20 patients with acute myocardial infarction. Infarction was anterior in 7 patients and inferior in 13. Patients with anterior infarction did not show ST segment depression in the inferior leads and all of the patients exhibited normal homogeneous accumulation of palmitate in the inferior and posterior walls. Nine patients with inferior infarction (69%) exhibited ST segment depression (apparently reciprocal or due to anterior wall ischemia) in the anterior precordial leads. Myocardial injury tended to be greater in the primary zone of necrosis among patients with inferior infarction and "reciprocal" ST segment depression compared with those without anterior ST segment depression. This was reflected by the greater total inferior ST segment elevation (0.48 ± 0.35 versus 0.07 ± 0.19 mV [± standard deviation], $p < 0.05$), peak plasma MB creatine kinase activity (354 ± 134 versus 80 ± 34 IU/liter, $p < 0.05$) and tomographically estimated infarct size (58 ± 13 versus 33 ± 10 PET-g-eq).

Three of the nine patients with inferior infarction and precordial ST depression exhibited anterior tomographic defects underlying the ST segment depression. Thus, although most of the patients with inferior infarction and precordial lead ST segment depression had no anterior wall metabolic compromise (67%) indicating that the anterior ST segment changes were truly reciprocal phenomena, in some the precordial electrocardiographic abnormalities reflected impaired metabolism in the anterior wall indicative of ischemia.

Acute transmural myocardial infarction is often accompanied by ST segment depression in leads thought to reflect electrical activity predominantly in sites distant from the identified zone of infarction. Interpretation of this phenomenon, often called reciprocal ST segment depression, remains controversial. Such electrocardiographic abnormalities may be indicative of ischemia or nontransmural infarction in regions distant from the primary infarct (1–3). Alternatively, they may represent electrical phenomena attributable exclusively to the primary locus (4–6). Differentiation of these two possible mechanisms has been attempted by correlative studies involving cardiac catheterization (1,3,6–8) and radionuclide ventriculography (4,9), which provide information about the functional integrity of the myocardium, and thallium scintigraphy (2,5). Unfortunately, although this procedure may provide qualitative estimates of myocardial perfusion, interpretation of results is complicated by variable attenuation and redistribution of the radioisotope and the use of a nonphysiologic tracer whose behavior is markedly influenced by transit time (10).

Positron emission tomography after the intravenous administration of carbon-11 palmitate permits quantitative assessment of regional myocardial metabolic integrity. We have previously shown (11–13) that carbon-11 palmitate is extracted avidly by normal myocardium and that regions of myocardial ischemia and infarction exhibit depressed accumulation of carbon-11 palmitate. Furthermore, the loci and extent of tomographically detectable decreased accumulation of carbon-11 palmitate correlate closely with the morphologic locus and extent of infarction in experimental

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0735-1097/83/$3 00

animals, the electrocardiographic site of infarction in patients and enzymatically estimated infarct size (13–15).

In the present study, positron tomography was utilized to determine whether patients with myocardial infarction who exhibit apparently reciprocal ST segment depression have abnormal metabolism in zones distant from the primary zone of infarction (accounting for the apparently "reciprocal" ST segment depression), or whether the ST changes are indeed reciprocal and not associated with abnormal metabolism in regions implicated electrocardiographically but distant from the primary zone of infarction.

Methods

Patient selection. Patients admitted to the cardiac care unit of Barnes Hospital with acute myocardial infarction documented by a typical history of chest pain and serial electrocardiographic abnormalities, and confirmed by characteristic serial changes in plasma creatine kinase and MB creatine kinase activity were included if the following criteria were met: 1) absent historical or electrocardiographic evidence of prior myocardial infarction; 2) lack of findings suggestive of primary valvular or heart muscle disease; 3) absence of manifestations of cardiogenic shock; 4) absence of conduction abnormalities that might preclude the electrocardiographic assessment of the locus of infarction (that is, left bundle branch block); 5) absence of a permanent electronic pacemaker; and 6) patient willingness to undergo positron emission tomography within 24 hours of the onset of symptoms. A total of 20 subjects met these criteria and were studied after written informed consent was obtained according to a protocol approved by the Human Studies Committee of Washington University on June 19, 1980. The patients included 16 men and 4 women with a mean age of 61 years (range 42 to 76).

Electrocardiographic evaluation. Electrocardiograms were recorded at a paper speed of 25 mm/s and a sensitivity of 1 mm = 0.1 mV. ST segment elevation and depression were measured relative to the TP segment 80 ms after the J point. Measurements from three beats were averaged for each lead studied. Significant ST segment depression was considered to be present if 0.1 mV depression or greater was present in at least two precordial leads (V_1 to V_6) or in at least two inferior leads (II, III, AVF). The total ST depression in six precordial leads or in the three inferior leads was then summed and expressed as total ST depression.

Acute transmural myocardial infarction was diagnosed when new Q waves of at least 30 ms duration developed in at least two anatomically adjacent leads. Patients not meeting this criterion were considered to have non-Q wave infarction. True posterior infarction was diagnosed according to the criteria of Perloff (16).

The initial 12 lead electrocardiogram was recorded 3.4 ± 3.3 hours (range 0.5 to 16, mean ± standard deviation) after the onset of chest pain. The electrocardiogram obtained most closely to the time of performance of positron emission tomography was used to classify patients according to the presence or absence of reciprocal ST segment depression. The interval between this electrocardiogram and the initial tomographic study averaged 3 ± 3.2 hours (range 0.25 to 10). Three of the 20 patients exhibited reciprocal ST segment depression on the initial electrocardiogram, which resolved before tomography. For the purposes of this study, these patients were classified as not having reciprocal ST segment depression. Nine patients exhibited persistent reciprocal ST segment abnormalities on an electrocardiogram recorded 2.7 ± 2.8 hours before tomography. In six of these nine patients, an electrocardiogram repeated shortly after tomography confirmed persistent ST segment depression. In the remaining three patients, a repeat electrocardiogram was not recorded until 18 ± 1.8 hours after tomography, at which time ST segment depression had resolved. Electrocardiograms were performed again at the time of repeat tomography before hospital discharge at a time when reciprocal ST segment depression had resolved in all patients in whom it had been present initially.

Additional evaluation. Total plasma creatine kinase and MB creatine kinase activity were measured at the time of patient admission, at least every 8 hours during the first 5 days of hospitalization and twice daily thereafter for the remainder of the hospital stay. Patients were observed for detection of potential complications of myocardial infarction. For the purposes of this study, complications were categorized as those reflecting congestive heart failure (diagnosed on the basis of rales that did not clear with a cough throughout at least the inferior half of the lung fields, pulmonary congestion documented by chest roentgenogram), extension of infarction (documented by a serial rise and fall in plasma MB creatine kinase activity after values had initially returned to baseline), persistent angina pectoris after the initial 24 hours of hospitalization, or death.

Tomographic methods. Carbon-11 palmitate was prepared by the addition of cyclotron-produced 11-carbon dioxide to magnesium bromide-pentadecane as previously described (17). After preparation and sterilization of the tracer by millipore filtration, 15 to 20 mCi of carbon-11 palmitate was injected intravenously into a peripheral vein. After a delay of at least 3 minutes to permit clearance of radioactivity from the blood pool, tomography was performed with a positron emission transaxial tomograph, which acquires data sufficient for simultaneous reconstruction of seven parallel transverse cross sections (slices) of the heart. After moving the patient 1 cm caudad and repeating the tomographic imaging procedure, a total of 14 interlacing transaxial slices were reconstructed, spanning a height of 12.35 cm. After a delay of at least 2 hours to permit decay of carbon-11 to background levels, 20 to 30 mCi of 11-carbon

monoxide was administered by inhalation to permit imaging of the cardiac blood pool with tracer amount of labeled carbon monoxide bound to hemoglobin. Total body radiation dose was less than 2 rem, and less than 7.5 rem was delivered to the blood. Imaging commenced between 3 and 5 minutes after the patient was given 11-carbon monoxide by inhalation so that clearance of radioactivity from the lungs would be complete. Patients were studied an average of 9 ± 7 hours (range 3.5 to 27) after the onset of chest pain and restudied an average of 14 ± 7 days (range 5 to 30) later.

Analysis of tomographic data. Tomographic reconstructions of the myocardial distribution of carbon-11 palmitate at the midventricular level in normal subjects exhibit homogeneous accumulation of tracer throughout a horseshoe-shaped region with a nearly constant apparent ventricular wall thickness. Patients with transmural infarction exhibit discrete regions of depression of accumulation of carbon-11 palmitate extending from the endocardial to epicardial surface, and patients with nontransmural infarction exhibit regions of apparent wall thinning and regions of spatially heterogeneous intramural depression of myocardial accumulation of carbon-11 palmitate (Fig. 1) (18).

Data were reconstructed in transverse, sagittal and coronal planes so that virtually all segments of the left ventricular wall could be visualized adequately. The orthogonal reconstructions were then displayed on a high resolution monitor (Conrac) in a 100 × 100 element grid with a 16 level gray scale. A printout was produced for each reconstruction of the region encompassing the heart with a Versatec printer/plotter in which a numerical value from 0 to 255 was assigned to each pixel, indicating the relative radioactivity detected within each corresponding region of tissue. All regions containing at least 50% of maximal myocardial radioactivity were considered normal and included in a contour constructed to encompass them. The residual myocardial region containing less than 50% of maximal myocardial radioactivity was considered abnormal (14). After superimposition of the data derived from the 11-carbon monoxide blood pool study on the reconstructions obtained after intravenous injection of carbon-11 palmitate, the endocardial borders of the abnormal zone were defined. The epicardial edge of the zone of metabolic compromise was then constructed assuming a uniform left ventricular wall thickness. The extent of metabolically compromised tissue was calculated by summing the number of pixels within all zones of depressed carbon-11 palmitate accumulation, with correction for residual radioactivity attributable to blood pool activity as previously described (18). Initial tomographic images were used to calculate the extent of infarction. Previous validation studies (unpublished observations) performed on another patient group in our laboratory revealed that calculations of infarct size observed tomographically by independent observers blinded to electrocardiographic and angiographic data correlated closely ($n = 14$, correlation coefficient [r] = 0.91, probability [p] < 0.001), that results of repeat studies after an average interval of 6 weeks (range 7 days to 5 months) in patients with completed infarction also correlated closely ($n = 10$ pair, $r = 0.91$, $p < 0.001$).

Transaxial, coronal and sagittal reconstructions were divided into eight regions for analysis of metabolic integrity of the myocardial walls (Fig. 2). A patient with anterior infarction would typically exhibit abnormalities in regions 1, 2, 3, 5 and 6. In contrast, abnormalities were typically seen in regions 4, 5, 7 and 8 in reconstructions acquired from a patient with inferior myocardial infarction. In the setting of an inferoposterior infarction, metabolic abnormalities in regions 1 and 2 were considered to be in loci distant from the locus of primary infarction. Conversely, in association with anterior infarction, tomographic abnormalities observed in regions 4, 7 and 8 were considered to be in loci distant from the primary locus of infarction. All tomographic studies were coded and interpreted by observers unaware of clinical, electrocardiographic or left ventricular function data.

Multigated blood pool imaging. In patients who did not require cardiac catheterization for clinical indications, ventricular performance and regional wall motion were assessed on the basis of radionuclide ventriculograms acquired

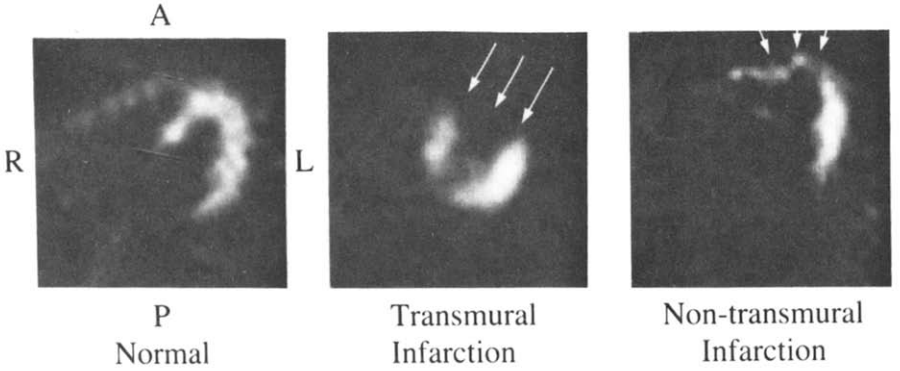

Figure 1. Positron-emission transaxial tomographic reconstructions of the heart at the midventricular level from a normal subject and from patients with transmural and nontransmural myocardial infarction performed after intravenous injection of carbon-11 palmitate. The **discontinuity** in the posterior aspect of the cardiac outline represents the relatively metabolically inactive mitral valve apparatus and atria. **Arrows** (↓) indicate regions of infarction. A = anterior; L = left; P = posterior; R = right.

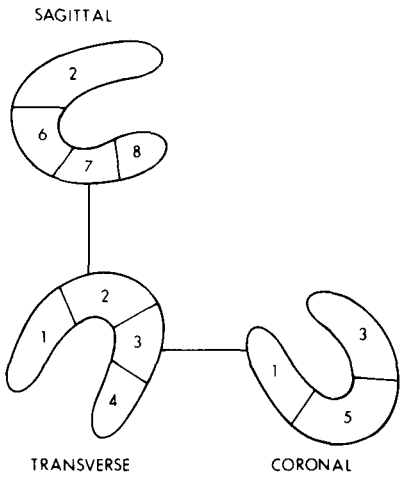

Figure 2. Schematic representations of transverse, coronal and sagittal reconstructions of the heart from a normal patient. 1 = septal wall; 2 = anterior wall; 3 = lateral wall; 4 = posterolateral wall; 5 = apical-inferior wall; 6 = apical wall, 7 = inferior wall; 8 = posterior wall.

with a mobile scintillation camera (Technicare—25.4 cm diameter, 0.63 cm thick sodium iodide crystal) after red blood cells had been labeled in vivo by the intravenous administration of 15.4 mg of stannous pyrophosphate followed 20 to 30 minutes later by intravenous injection of 20 to 30 mCi of technetium-99m pertechnetate. Anterior, 30 to 45° and 70° left anterior oblique projections were obtained. Wall motion was analyzed subjectively by the consensus of at least two observers who were unaware of the results of tomography and electrocardiographic findings. Left ventricular ejection fraction was calculated from a region of interest in the left anterior oblique projection as previously reported and validated (19). Each left ventricular wall was scored from +3 (normal) to −1 (dyskinetic) as previously described (20). Radionuclide ventriculograms were acquired an average of 5 days (range 1 to 13) after the apparent onset of infarction.

Cardiac catheterization. Sixteen patients underwent left ventriculography and selective coronary arteriography. Thirteen of these 16, who were considered for aggressive medical or surgical therapy, were studied angiographically during the acute phase of myocardial infarction, immediately after the initial tomographic study; 3 were studied between 47 and 181 days after infarction. Maximal luminal diameter narrowing for each major coronary artery was evaluated by observers unaware of the results of tomography. Narrowing in the diagonal or marginal branches was considered to be a lesion of the left anterior descending or circumflex coronary system, respectively. Stenoses greater than 50% of luminal diameter were considered significant.

Single plane right anterior oblique and left anterior oblique contrast ventriculograms were analyzed subjectively for regional wall motion abnormalities by observers blinded to the results of tomography and electrocardiographic findings. Hypokinesia was defined as mild to moderate impairment of regional systolic inward motion, akinesia as the absence of regional systolic motion and dyskinesia as paradoxical systolic expansion. For analysis of regional wall motion, the ventriculogram acquired in the right anterior oblique projection was divided into five segments (anterobasal, anterolateral, apical, diaphragmatic and posterobasal) and the ventriculogram acquired in the left anterior oblique projection was divided into two segments (septal and posterolateral) as described by Austin et al. (21). Ejection fraction was calculated from the right anterior oblique projection as previously described (22).

Statistical analysis. Data are reported as mean ± standard deviation. Differences between the means of independent continuous variables were assessed with a two-tailed Student's *t* test. Differences between discrete variables were assessed by chi-square analysis (with Yates' correction).

Results

Patients With Anterior Infarction

Among the 20 patients studied, 7 had anterior infarction and 13 had inferior infarction (Table 1). Reciprocal ST segment depression was not observed in the inferior leads in any of the seven patients with anterior infarction. Six of these seven patients had transmural myocardial infarction. Left ventricular ejection fraction ranged from 0.26 to 0.54 (0.45 ± 0.09), and peak plasma MB creatine kinase values ranged from 47 to 540 (259 ± 186 IU/liter). Five patients underwent cardiac catheterization at the time of infarction and one within 30 days. All seven patients exhibited regions of depressed accumulation of carbon-11 palmitate limited to the septum, anterior and apical walls. Although the apex was involved generally, no metabolic abnormalities were evident in the inferior and posterior walls. Repeat tomography showed no new regions of depressed accumulation of palmitate. Contrast and radionuclide left ventriculography demonstrated regional wall motion abnormalities limited to the anterior wall, septum and apex with no wall motion abnormalities in the inferior or posterior walls.

Significant obstructive disease of the left anterior descending coronary artery was observed in all patients studied angiographically. In addition, one patient had significant narrowing of the right coronary artery and three had significant obstructions in the circumflex system without concomitant metabolic abnormalities detectable tomographically in the myocardial segments supplied by these vessels.

Patients With Inferior Infarction

Inferior infarction with precordial lead ST depression. Nine (69%) of the 13 patients with inferior infarction exhibited ST segment depression (reciprocal or due to an-

Table 1. Clinical Characteristics of Patients

A. Patients With Anterior Myocardial Infarction

Case	Age (yr) & Sex	Infarct Type	Inferior Wall Defect by Positron Tomography	Inferior Wall Motion Abnormality	RCA or LCx Lesion
1	54M	TM	None	None	3+ RCA; 5+ LCx
2	56M	TM	None	None	None
3	76F	TM	None	None	3+ LCx
4	64M	TM	None	None	None
5	61M	TM	None	None	3+ LCx
6	50M	NTM	None	None	Not studied
7	69M	NTM	None	None	None

B. Patients With Inferior Myocardial Infarction With "Reciprocal" Changes

Case	Age (yr) & Sex	Infarct Type	Anterior Wall Defect by Positron Tomography	Anterior Wall Motion Abnormality	LAD or LCx Lesion
8	57M	TM	Present	Anterobasal	3+ LAD/5+ LCx
9	53M	TM	Present	Anterolateral	3+ LAD/5+ LCx
10	63M	TM	Present	Anteroseptal	5+ LAD/4+ LCx
11	49M	TM	None	None	None
12	60F	TM	None	None	None
13	66F	TM	None	None	4+ LAD/5+ LCx
14	66M	TM	None	None	5+ LAD/3+ LCx
15	57M	TM	None	None	Not studied
16	43M	TM	None	None	3+ LAD
17	65M	TM	Present	None	1+ LAD
18	76M	TM	None	None	Not studied
19	52F	NTM	None	None	Not studied
20	66M	TM	None	None	2+ LAD

F = female; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; M = male; NTM = nontransmural myocardial infarction; RCA = right coronary artery; TM = transmural myocardial infarction; 1+ = 0–4%; 2+ = 25–49%; 3+ = 50–74%, 4+ = 75–99%; 5+ = 100% reduction in coronary artery luminal diameter.

terior wall ischemia) in the anterior precordial leads on the electrocardiogram recorded immediately before tomography. The inferior infarction was transmural in each of these nine patients. The sum of the extent of ST elevation in the three inferior leads averaged 0.48 ± 0.35 mV. The total extent of ST segment depression in the precordial leads averaged 0.86 ± 0.41 mV. One patient in this group had initial true posterior infarction confirmed by cardiac catheterization and coronary angiography. Another patient developed true posterior myocardial infarction during the early hospital course. Congestive heart failure developed during the cardiac care unit phase of infarction in three patients and persistent postinfarction angina occurred in one. One patient died during the acute phase of infarction. Peak plasma MB creatine kinase activity averaged 254 ± 134 IU/liter (range 23 to 432). Left ventricular ejection fraction was 0.51 ± 0.12 (range 0.31 to 0.63). Cardiac catheterization was performed in eight patients and within 24 hours of the onset of infarction in six.

Positron tomography. Among the nine patients with inferior myocardial infarction and anterior precordial ST segment depression, decreased accumulation of carbon-11 palmitate was evident in the inferior, posterior, apical and posterolateral left ventricular walls corresponding to the electrocardiographically localized primary zone of infarction. Three patients exhibited additional regions of depressed accumulation of carbon-11 palmitate in the septum and anterior walls corresponding to ischemic regions potentially accounting for ST segment depression (Fig. 3). In two of these three patients, partial restitution of the accumulation of carbon-11 palmitate was evident in the anteroseptal walls at the time of repeat tomographic study when reciprocal ST segment depression had resolved. One patient (Case 10) showed persistent depressed accumulation of palmitate in the anterior wall on repeat tomography.

Coronary angiography. Multivessel coronary artery disease with significant obstruction of the left anterior descending coronary artery was observed in each of the three patients

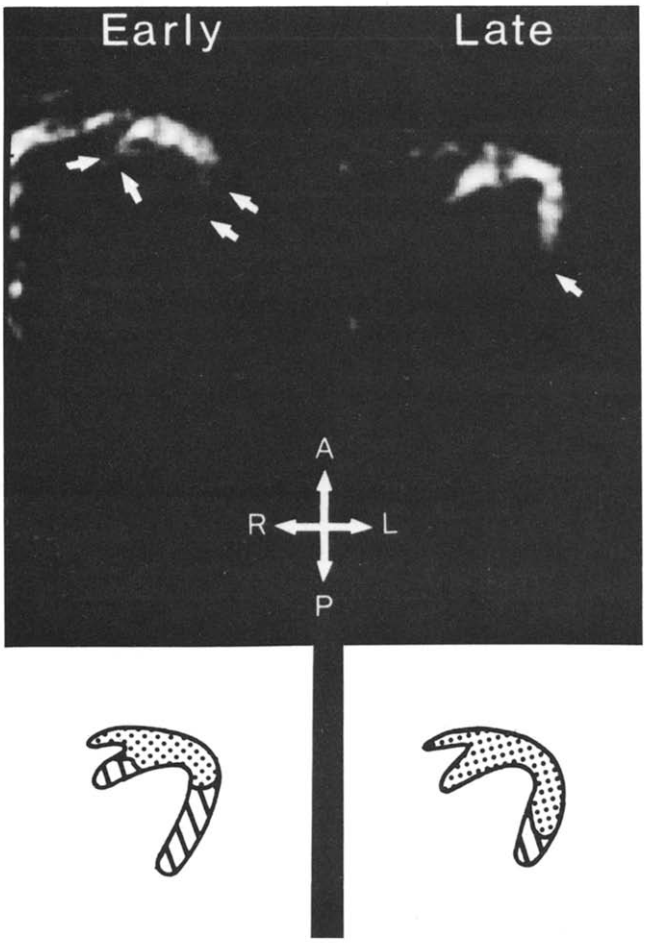

Figure 3. Representative midventricular transverse reconstruction from a patient with inferior myocardial infarction and anterior precordial ST segment depression obtained with positron tomography after carbon-11 palmitate acquired within the first 10 hours after infarction (**upper left**) and 13 days later (**upper right**). Schematic representations are shown below. The **stippled areas** indicate regions of normal accumulation of carbon-11 palmitate and the **slashed areas** indicate regions of depressed accumulation. The initial study shows depressed accumulation of carbon-11 palmitate in the septum and posterolateral walls (**arrows**). At the time of repeat study, some restitution of accumulation of palmitate was evident within the septum and lateral wall. Abbreviations as in Figure 1.

with depressed accumulation of palmitate in the anterior and septal walls. Among six patients with normal accumulation of palmitate in the anterior and anteroseptal walls, coronary obstructive lesions were confined to the right coronary artery in two, the left anterior descending coronary artery and right coronary artery in one, and two patients showed severe three vessel obstructive disease. One patient did not undergo cardiac catheterization. Thus, among the nine patients with inferior myocardial infarction and ST segment depression in the anterior precordial leads, three patients (33%) exhibited depressed accumulation of palmitate in the anterior and septal walls and had significant obstructive disease in the left anterior descending coronary artery. Each of these three patients had an abnormality of left ventricular regional wall motion detected by contrast or radionuclide ventriculography that corresponded to the locus of depressed accumulation of palmitate. All of the remaining six patients exhibited normal myocardial metabolism in the anterior left ventricular wall and normal segmental left ventricular wall motion in the regions distant from the inferior infarction, despite significant obstruction of the left anterior descending coronary artery in three patients.

Inferior infarction without precordial lead ST depression. Among the four patients with inferior myocardial infarction without precordial ST segment depression, infarction was apparently transmural in three, and true posterior infarction was present in one. An extension of infarction occurred on the 11th day in one patient. No other complications were observed. The mean total ST segment elevation was 0.07 ± 0.19 mV, significantly less than that observed among patients with inferior infarction and anterior precordial ST segment depression. Peak MB creatine kinase averaged 79.5 ± 33.7 IU/liter among the four patients with inferior infarction without anterior lead ST segment depression, significantly less than that observed among the nine patients with inferior infarction and significant precordial lead ST segment depression ($p < 0.05$) (Fig. 4). Left ventricular ejection fraction averaged 0.58 ± 0.17.

Positron tomography. Significant regional depression of the accumulation of carbon-11 palmitate was present in the inferoposterior left ventricular wall in each of these four patients. Repeat tomography again demonstrated depressed accumulation of palmitate confined to the inferoposterior left ventricular wall. Mean extent of myocardial metabolic compromise, estimated from the initial tomographic study, tended to be compared less with values in patients with inferior infarction associated with precordial lead ST segment depression (33 ± 10 versus 58 ± 13 PET-g-Eq). A small anterior region exhibiting depression of accumulation of palmitate was present in one patient in this group (Case 17, Table 1), which resolved by the time of repeat study (on Day 10), and was associated with less than 50% obstruction in the left anterior descending coronary artery, which may represent a false positive result. No patient in this group exhibited anteroseptal wall motion abnormalities. No new areas of depressed accumulation of palmitate were noted on repeat tomographic study in any patient in this group.

Discussion

Previous studies on reciprocal ST depression in acute myocardial infarction. The implications of apparently reciprocal ST depression in acute myocardial infarction have not been thoroughly elucidated. In experimental animals

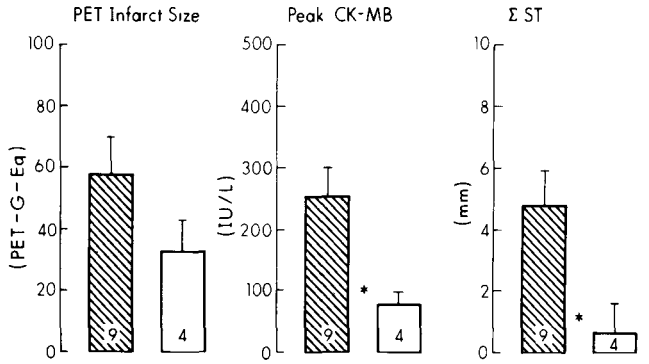

Figure 4. Relation between the extent of myocardial damage and the presence of precordial ST segment depression in patients with inferior myocardial infarction assessed by positron emission tomography (PET-g-Eq), peak plasma MB creatine kinase (CK-MB) (IU/liter) and total (Σ) ST segment elevation (mm). **Slashed bars** indicate patients with anterior precordial ST depression and **open bars** indicate patients without precordial ST segment depression (*$p < 0.05$).

with transmural injury produced by coronary artery ligation, ST elevation is evident in electrodes subserving the area of injury. Simultaneous ST depression appears in epicardial leads in regions from the locus of infarction (23–25). ST segment depression also may be seen in zones surrounding the central ischemic regions (26). Autopsy studies (27,28) of patients with fatal acute inferior myocardial infarction associated with ST depression in the precordial leads generally show an absence of infarction of the anterior wall, and the precordial lead ST segment depression has been judged to be reciprocal to posterior or posterolateral infarction.

Patients with inferior myocardial infarction often manifest significant stenosis of the left anterior descending coronary artery (29,30) in addition to critical obstruction of the right coronary artery. This observation has led to the view that occult disease in the left anterior descending artery may become manifest as precordial ST depression due to the hemodynamic stress of an inferior myocardial infarction (1–3,31) and the effects of high circulating catecholamines (3,5), reduced collateral blood flow from the occluded right coronary artery (5) and spasm in the left anterior descending artery (3) on anterior wall oxygen demand and supply. However, little direct evidence has been acquired suggesting that the region of myocardium implicated by the precordial lead ST segment depression is potentially ischemic.

Significance of precordial ST depression in inferior wall infarction. Our results indicate that true electrocardiographically reciprocal ST depression is common in patients with inferior myocardial infarction (69%), consistent with an apparent incidence reported by others (1–9,31,32) averaging 57%. Patients exhibiting this phenomenon tend to manifest more extensive transmural infarction and involvement of the posterior wall (4,5,31), greater ST elevation in the inferior leads (5,7,9), a higher incidence of clinical complications (1,31) and higher peak values of plasma MB creatine kinase activity (4,5,31) compared with findings in patients without reciprocal ST depression. Although coronary artery spasm has been reported (33) in some patients catheterized during the acute phase of myocardial infarction, we did not observe spasm of the left anterior descending coronary artery in patients with inferior myocardial infarction at a time when reciprocal ST depression was present. This confirmed the results of others (6–8). On the other hand, three (33%) of the nine patients with inferior infarction and precordial ST segment depression had ischemia affecting myocardium distant from the site of inferior infarction, which was reflected by results of positron emission tomography. It should be noted that this conclusion is based on a small number of patients.

Therefore, although most of the patients we studied with inferior infarction and precordial lead ST segment depression had no anterior wall compromise (67%) and thus exhibited true electrocardiographically reciprocal ST depression with or without significant left anterior descending coronary artery obstructive disease, in some the electrocardiographic change reflected impaired metabolism in the anterior wall indicative of ischemia. The two types of ST segment depression could not be differentiated simply by electrocardiographic assessments.

Patients with inferior infarction without precordial lead ST depression generally exhibited normal, homogenous accumulation of carbon-11 palmitate in the anterior left ventricular wall, even in the presence of angiographically significant obstruction of the left anterior descending coronary artery. One patient in this group manifested transient, modest, anterior regional depression of accumulation of palmitate.

Significance of reciprocal ST depression in anterior wall infarction. Despite reports (9) of reciprocal ST changes in many patients with anterior infarction, none of our patients with anterior infarction exhibited apparently reciprocal ST depression in the inferior electrocardiographic leads. The differing observations between our group and those previously reported may well be due to the relatively small sample size of our group. In all of our patients, accumulation of carbon-11 palmitate was homogeneous in the inferoposterior left ventricular wall. Inferior regional wall motion was normal.

Clinical correlates of anterior ST depression in inferior infarction. The results obtained in this study are compatible with those of others, suggesting that anterior ST segment depression associated with inferior infarction may represent either true electrical reciprocal change or ischemia of anterior wall myocardium. Anterior wall motion abnormalities have been documented by radionuclide ventriculography (2,4,5,31) and anterior myocardial perfusion defects have been observed with thallium scintigraphy (2,5) in some, but not all, patients with inferior myocardial infarction and precordial lead ST depression. Bush et al. (2)

have shown that patients with inferior infarction, reciprocal ST depression and ischemia in the anterior myocardial wall had a higher incidence of postinfarction angina than patients with precordial lead ST depression without independent evidence of anterior wall ischemia.

Clinical implications. The present identification of anterior wall injury or ischemia in a subset of patients with inferior infarction and precordial lead ST depression complements data provided by coronary arteriography (1,3) because the presence of left anterior descending obstructive lesions does not necessarily imply that the anterior myocardium will manifest the metabolic sequelae of ischemia, even at the time of precordial lead ST depression. Our findings indicate that apparently reciprocal ST depression is quite common in patients with inferior myocardial infarction. When patients with inferior infarction with apparently reciprocal ST depression are studied at the time of infarction by positron emission tomography with carbon-11 palmitate, most show no evidence of anterior wall depression of accumulation of carbon-11 palmitate, whether or not angiographically significant obstruction is present in the left anterior descending system. However, some patients with apparently reciprocal ST depression manifest depressed accumulation of palmitate anteriorly as well as anterior wall motion abnormalities. Thus, the apparently reciprocal ST depression in such patients appears to be a reflection of ischemia of the anteroseptal wall. It should be noted that our study group was small and these conclusions could be affected by the small sample size. The identification of additional regions of myocardium at risk in patients with acute inferior infarction is particularly important because patients with ischemia at a distance from the primary zone of infarction have a poor short-term prognosis (34) and may, therefore, be particularly suitable candidates for prompt and aggressive therapy designed to protect jeopardized myocardium.

We thank Michael Welch, PhD, Carmen Dence, David R Marshall and William Morgenau for the preparation of carbon-11 palmitate, Theron Baird and Elizabeth Galie for their technical assistance and Joyce Kalayeh for her help in preparing the typescript

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